[30] References here to "MTb" refer to *Mycobacterium tuberculosis*. The sequence of the entire genome of MTb is set forth in TubercuList, which can be found on the Internet by typing "http://" followed by "genolist.pasteur.fr/TubercuList/."

Please delete paragraph [62] and insert in its place:

[62] In a particularly preferred embodiment, the EtaA gene can be amplified using the primers 5'-GGGGTACCGACATTACGTTGATAGCGTGGA-3' (SEQ ID NO:3) and 5'-ATAAGAATGCGGCCGCAACCGTCGCTAAAGCTAAACC-3' (SEQ ID NO:4) (EtaA). Many other primer sets can be selected using standard programs widely available in the art. For example, the program "Primer3" can be found on-line by typing "www-" followed by "genome.wi.mit.edu/cgi-bin/primer/primer3_www.cgi." This program was used to select the primer pairs noted above, using the default conditions. The program was also used to select the following sequencing primers, which can be used to amplify sections of the EtaA gene for sequencing:

5' ATCATCCATCCGCAGCAC 3' (SEQ ID NO:5);
5' AAGCTGCAGGTTCAACC 3' (SEQ ID NO:6);
5' GCATCGTGACGTGCTTG 3' (SEQ ID NO:7);
5' AAGCTGCAGGTTCAACC 3' (SEQ ID NO:8);
5' TGAACTCAGGTCGCGAAC 3' (SEQ ID NO:9);
5' AACATCGTCGTGATCGG 3' (SEQ ID NO:10);
5' ATTTGTTCCGTTATCCC 3' (SEQ ID NO:11);
5' AACCTAGCGTGTACATG 3' (SEQ ID NO:12);
5' TCTATTTCCCATCCAAG 3 (SEQ ID NO:13); and
5' GCCATGTCGGCTTGATTG 3' (SEQ ID NO:14).

Please delete paragraph [81] and insert in its place:

[81] The production of metabolite (**5**) from ETA by tuberculosis is surprising as 4-pyridylmethanol is a major metabolite of INH by whole cells of MTb (Youatt, J. *Aust J Chem* 14:308 (1961); Youatt, J. *Aust J Exp Biol Med Sci* 38:245 (1960); Youatt, J. *Aust J Biol Med Sci* 40:191 (1962)). Like spontaneous oxidation of INH, spontaneous oxidation of ETA fails to produce any trace of the major *in vivo* metabolite, (2-ethyl-pyridin-4-yl)methanol. INH has been shown to be activated by KatG *in vitro* to a variety of products including isonicotinic acid, isonicotinamide and isonicotinaldehyde (which *in vivo* is rapidly reduced to 4-pyridylmethanol) (Johnsson, K. et al., *J Am Chem Soc* 116:7425 (1994)). INH metabolism to 4-pyridylmethanol only occurs in drug-susceptible organisms while drug-resistant organisms no longer produce this metabolite (Youatt, J., *Am Rev Respir Dis* 99:729 (1969)). Similarly, we postulate that ETA is activated via the corresponding S-oxide to a sulfinate that can form an analogous aldehyde equivalent (an imine) through a radical intermediate (Paez, O.A. et al., *J Org Chem* 53:2166 (1988)).

Please delete paragraph [88] and insert in its place:

[88] INH (**6**) has been shown to be activated by KatG in vitro to a variety of products including isonicotinic acid, isonicotinamide and isonicotinaldehyde (**9**) (which *in vivo* is rapidly reduced to 4-pyridylmethanol (**10**)) (Johnsson, K. & Schultz, P. G., J Am Chem Soc 116:7425-68 (1994)). The results support the notion that in vivo INH is metabolized by oxidation to an acyl diimide (**7**), then to a diazonium ion (**8**) or an isonicotinyl radical which may abstract a hydrogen atom from a suitable donor to form isonicotinaldehyde. Similarly, we postulate that ETA is activated via the corresponding S-oxide (**2**) to a sulfinate that can form an analogous aldehyde equivalent (an imine) through a radical intermediate. Hydrolysis of this imine could be followed by reduction of the resulting aldehyde to the observed metabolite (**5**).